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A M E R I C A N C O L L E G E O F



P H Y S I C I A N S[®]

Nocturnal Efficiency and Tolerance of a Demand Oxygen Delivery System in COPD Patients With Nocturnal Hypoxemia*

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Objectives: We compared the efficacy of the standard nasal cannula and the demand oxygen delivery system (DODS) during sleep in patients with COPD.

Subjects: Twenty patients with moderate or severe COPD were included in the study.

Methods: Four consecutive polysomnographic recordings were performed under the following conditions: DODS powered by compressed air (night 1 [N1]); oxygen delivered with a nasal cannula alone (night 2 [N2]); oxygen delivered through a DODS (night 3 [N3]); and oxygen delivered with nasal cannula alone (night 4 [N4]). Oxygen flow rates with and without DODS were adjusted the day before the first night so that the resulting transcutaneous arterial oxygen saturation (SaO_2) was $\geq 95\%$. The following parameters were evaluated each night: apnea-hypopnea index, nocturnal SaO_2 , total oxygen saving, and several neurophysiologic parameters.

Results: The oxygen saving with the DODS was, on average, 60%. All parameters obtained during N2 and N4 (oxygen alone) were identical. The percentage of total recording time spent at $SaO_2 \geq 95\%$ was comparable between N2 ([mean \pm SD]; $69 \pm 32\%$) and N3 ($61 \pm 31\%$) (difference is not significant [NS]), as was the time spent at SaO_2 between 90% and 95% (N2, $29.8 \pm 31\%$; N3, $35.9 \pm 27\%$; NS) and $< 90\%$ (N2, $0.75 \pm 2.6\%$; N3, $2.5 \pm 8.6\%$; NS). Although the mean response time was not significantly different between N2 and N3, two patients experienced a substantial increase in response time with an $SaO_2 < 90\%$ on the DODS. The DODS device did not induce any difference in the percentage of time spent in rapid eye movement (REM) sleep (N2, $12.3 \pm 8.7\%$; N3, $16.4 \pm 7.8\%$; NS) or non-REM sleep (N2, $87.7 \pm 8.7\%$; N3, $83.7 \pm 7.9\%$; NS). Non-REM distribution in stage 1–2 sleep and in stage 3–4 sleep was comparable between N2 and N3. Similarly, no difference was observed for the sleep efficiency index (N2, $71 \pm 15\%$; N3, $69.6 \pm 14\%$; NS). Differences between sleep onset latency times were NS.

Conclusions: In a majority of moderate to severe COPD patients, the use of a DODS device does not induce any significant alteration of nocturnal neurophysiologic and ventilatory profiles. However, the presence of nocturnal desaturation in a few patients justifies the need to systematically perform a ventilatory polygraphic recording when prescribing a DODS device. (CHEST 1999; 116:22–29)

Key words: COPD; demand oxygen delivery system; nocturnal hypoventilation; oxygen therapy

Abbreviations: AHI = apnea-hypopnea index; AI = apnea index; DODS = demand oxygen delivery system; LTO = long-term oxygen therapy; NREM = non-rapid eye movement; NS = not significant; REM = rapid eye movement; SaO_2 = arterial oxygen saturation; SOL = sleep onset latency; TST = total sleep time

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Because long-term oxygen therapy (LTO) administered > 5 h/d has led to increased survival in hypoxemic COPD patients,^{1,2} it could be assumed that correction of hypoxemia during rapid eye movement (REM) and non-REM (NREM) sleep is beneficial in the management of these patients. Oxygen availability during the night and during the day has been greatly improved with concentrators, but these

devices are noisy and remain stationary. In contrast, administration of ambulatory oxygen is a mainstay procedure to augment daily duration of LTO, thus improving treatment efficacy³ either with compressed or liquid oxygen cylinders. To deliver an optimal amount of oxygen during a 24-h period, liquid oxygen therapy has recently been developed; and, currently, portable gas cylinders may be obtained from a compressor linked to a concentrator. The use of these ambulatory devices raises the issue of oxygen preservation either from an economic point of view or to maximize patient autonomy.

Demand oxygen delivery system (DODS) devices have been designed to increase oxygen autonomy with gas or liquid portable reservoirs. DODS devices deliver oxygen only during the inspiratory phase of the respiratory cycle and, therefore, permit oxygen leaks to be kept at a minimum and reduce oxygen costs. Because the use of a portable oxygen source (gas or liquid) is associated with a better compliance with LTO,³ previous studies have focused on the efficiency of DODS devices during ambulatory activities.⁴⁻⁶ However, consequences of the use of DODS devices on sleep quality and quantity in patients with moderate or severe COPD should also be assessed.

Therefore, we designed a study to compare respiratory pattern and sleep parameters in COPD patients using a liquid oxygen reservoir with and without a DODS device.

MATERIALS AND METHODS

Twenty consecutive patients with stable COPD who presented with major obstructive syndrome and chronic respiratory insufficiency gave their informed consent to participate in the study. Twelve of these patients were receiving LTO before entering the study. No patient had any clinical sign suggesting an associated sleep apnea syndrome.

The DODS that was used (Optimox; Taema; Antony, France) is a device that only delivers oxygen during the inspiratory phase. An inspiration pressure (\pm SD) of -2 ± 0.5 Pa is sufficient to trigger the system. The oxygen flow rate is determined by a switch that permits modification of the oxygen volume delivered during each inspiration. The valve is equipped with an apnea card designed to detect apneas lasting more than 10 ± 2 s. In this instance, the device delivers a continuous oxygen flow rate lasting 18 ± 2 s.

During the afternoon preceding the first recording night, arterial blood gas contents measurement on room air, respiratory function tests, full blood count, and chest radiograph were performed. Oxygen titration was assessed with the patient in a supine position, resting and awake, by using continuous monitoring of the transcutaneous oxygen saturation (Biox 3700; Ohmeda; Louisville, CO). Oxygen was delivered by a liquid reservoir (Freelox; Taema), and flow rates were fitted after successive tests, each lasting 1 h, so that the arterial oxygen saturation (SaO_2) was $\geq 95\%$. The adjustments were first performed with oxygen alone and then with oxygen and the DODS device (oxygen + DODS). Four consecutive polysomnographic recordings were made and administered to

the patient according to a single-blind methodology. The first night (N1) was considered an adjustment night allowing the patient to adapt to the DODS and the various transducers, with an attempt to eliminate the first-night effect. The patient was then connected to a compressed air supply driving the DODS device. During the second night (N2), polysomnography was performed by using oxygen without DODS (oxygen alone) at the flow rate defined on the first day, ensuring an $\text{SaO}_2 \geq 95\%$. During the third night (N3), the patient was given oxygen with a DODS (DODS + oxygen). To assess and control the quality of oxygenation, recording conditions during the fourth night (N4) were the same as those during N2 (oxygen alone). Quality of recordings and oxygen therapy compliance were assessed during all four nights by a qualified technician.

Oxygen consumption during N2, N3, and N4 was estimated by the weight difference of the Freelox liquid oxygen reserve between the previous evening and the morning after each polysomnographic recording. Weight measurements were assessed with a precision (± 1 g) scale (model E/3; Sauter; Hightstown, NJ).

Respiratory parameters were measured with a monitor (Respi-somnograph; Nellcor Puritan Benett; Antony, France) and an oximeter (Biox 3700; Ohmeda). These parameters included transcutaneous finger pulse oximetry, electrocardiogram, thoracoabdominal movements, and nasobuccal airflow measured by thermistors at the nose and mouth. The automated analysis was checked and corrected on the monitor screen by the same qualified physician. Apnea was defined as an interruption of nasobuccal air flow lasting at least 10 s that was subsequently classified as obstructive, central, or mixed. Hypopnea was defined as a reduction of the amplitude of nasobuccal air flow by at least 50% associated with a fall in SaO_2 of $\geq 4\%$.

Sleep parameters were determined according to the criteria established by Rechtschaffen and Kales.⁷ Neurophysiologic signals were recorded simultaneously by an Oxford Medilog 9000 (Oxford Instruments Sarl; Orsay, France), including an electromyogram, an electro-oculogram, and an EEG with frontal, vertex, occipital, median, and ocular electrodes. Data were analyzed in a computer-assisted manner followed by repeat reading of the raw data by a neurophysiologist who was not aware of the sequence of nights. The recording EEG cassettes were analyzed in 60-s epochs to determine total sleep time (TST); REM and NREM sleep duration; REM latency; sleep onset latency (SOL), defined as the time from lights out to the occurrence of the first stage 2 sleep; and sleep efficiency, defined as the ratio between TST and total time spent in bed. Arousals were determined according to standard criteria⁷ and defined as awake periods lasting at least 60 s.

Statistical Analysis

Data were analyzed with a statistical software package (Stat-View; Abacus Concepts; Berkeley, CA). Tests of normality were performed on EEG indices. Student's *t* tests were used for comparisons of variables with normal distributions. Otherwise, between-group comparisons were performed using a Wilcoxon paired test or a nonparametric Friedman's test. The level of significance was set at 5%. For all parameters, we did not find any statistical difference between the two oxygen nights, N2 and N4.

RESULTS

Patient Characteristics

The study included 20 patients, 18 men and 2 women, with a mean age of 60.4 ± 8 years (Table 1).

Table 1—Characteristics of Study Subjects*

Subject No.	Age, yr	Sex	BMI, kg/m ²	PaO ₂ , mm Hg	PaCO ₂ , mm Hg	VC, L (%)	FEV ₁ , L (%)	FEV ₁ /VC, %	RV/TLC, %
1	56	M	16.76	51.7	49.8	1.53 (41)	0.92 (32)	60	164
2	54	M	21.97	58.6	48.4	2.52 (60)	0.84 (26)	33	141
3	69	F	30.7	54.9	52.4	1.15 (55)	0.4 (24)	34.7	122
4	62	M	17.99	57.0	45.8	1.85 (50)	0.76 (27)	41.1	170
5	47	M	29.41	57.9	54.3	2.1 (48)	0.92 (27)	43.8	192
6	59	M	22.69	57.1	42.7	2.27 (47)	1.04 (28)	45.8	153
7	53	M	21.93	57.1	51.2	1.52 (39)	0.8 (26)	52.6	121
8	47	M	20.32	63.2	36.1	1.8 (47)	1 (32)	55.5	NA
9	66	M	25.26	46.6	51.6	1.43 (35)	0.64 (21)	44.7	179
10	70	M	26.58	48.0	47.6	2.95 (64)	1.12 (33)	37.9	138
11	58	F	23.23	50.4	42.9	1.83 (68)	0.72 (33)	39.3	154
12	72	M	24.16	57.8	43.6	1.96 (45)	0.84 (25)	42.8	125
13	56	M	29.05	58.5	46.3	2.77 (70)	1.8 (53)	65	168
14	60	M	19.8	48.0	52.3	1.61 (43)	0.68 (24)	42.2	190
15	71	M	25.4	63.2	51.6	1.43 (34)	0.6 (20)	41.9	165
16	70	M	19.1	42.0	53.7	1.04 (30)	0.64 (25)	61.5	201
17	61	M	24.02	58.5	51.4	2.35 (63)	1.04 (36)	44.2	147
18	58	M	24.33	54.7	44.0	2.96 (70)	1.16 (36)	39.2	111
19	69	M	32.18	58.6	52.6	1.99 (52)	1.32 (40)	66.3	137
20	50	M	23.87	54.1	42.3	2.56 (65)	0.76 (24)	29.3	176
Mean	60.4		23.94	54.9	48.03	1.98 (51.30)	0.90 (29.6)	46.04	155.47
± SD	8.04		4.17	5.54	4.89	0.57 (12.54)	0.30 (7.67)	10.64	26.09

*BMI = body mass index; NA = not available; M = male; F = female.

All patients had moderate to severe stable COPD, with a predicted mean FEV₁ of 29.6 ± 7.6%, a predicted mean FVC of 51.3 ± 12.5%, and a predicted mean residual volume to total lung capacity ratio of 155.47 ± 26.09%. No subject had any clinical sign suggesting an associated sleep apnea syndrome. Mean body mass index was 23.94 ± 4.17 kg/m². Mean PaO₂ at rest was 54.9 ± 5.54 mm Hg, and most patients had hypercapnia (mean PaCO₂ = 48.03 ± 4.89 mm Hg). Two patients had a PaO₂ value > 60 mm Hg in room air and in stable state, but they were included in the study. The first patient was clinically unstable for many years, experiencing dyspnea with clinical signs, ECG, and chest radiographs indicating right heart failure. On entering the study, this patient was treated by ambulatory liquid oxygen because of significant desaturations with exercise. The second patient had been hypercapnic for several years and had a typical COPD pattern with frequent bronchospastic exacerbations.

All subjects were current or ex-smokers with a smoking history of > 10 pack-years. In room air (N1), apnea index (AI) was 2.1 ± 1.6 events/h and the apnea-hypopnea index (AHI) was 4.8 ± 4.3 events/h. Oxygen supplementation with or without DODS did not induce any significant increase of nocturnal apneas or hypopneas (Fig 1) at N2 (AI, 2.1 ± 1.8 events/h; AHI, 4.9 ± 4.0), N3 (AI, 2.3 ± 2.0; AHI, 4.5 ± 3.4), or N4 (AI, 2.3 ± 1.4; AHI, 5.0 ± 3.9).

Oxygen Saving With the DODS

To achieve a transcutaneous SaO₂ of > 95%, oxygen flow rates were regulated slightly higher when using a DODS device than with nasal cannulas alone (respectively, 3.0 ± 1.4 L/min and 2.4 ± 0.9 L/min). The mean oxygen consumption was 1.669 kg for N2 and 1.379 kg for N4. Use of a DODS device permitted highly significant saving because oxygen consumption was 0.573 kg for N3 (p = 0.003 comparing N2 with N3), which represents approximately 60% reduction in oxygen consumption (Fig 2).

Nocturnal SaO₂ Using Oxygen With or Without the DODS

With DODS + room air (N1), all 20 patients spent a mean of 52 ± 35.2% (extremes, 0% to 100%) of total recording time with an SaO₂ of < 90%. When receiving oxygen, 18 patients spent 100% of the night with an SaO₂ level of ≥ 90%, whether the DODS was used (N3) or not (N2) (Fig 3). Two patients (Nos. 1 and 18) who spent only 1% of the night with an SaO₂ level of < 90% when receiving oxygen (N2 and N4) had a worsened nocturnal desaturation when given DODS + oxygen (N3), with 13% and 37% of the night, respectively, spent with an SaO₂ level of < 90%. Both patients were characterized as having rather low PaO₂ levels in

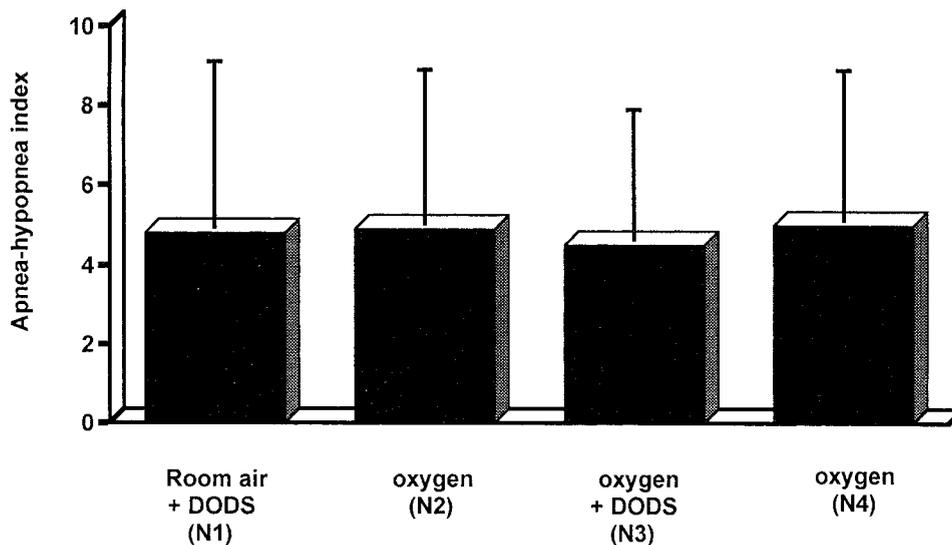


FIGURE 1. Values of AHI calculated from total recording time. No statistical differences were found between the values recorded for each night.

room air. However, they had no greater pulmonary function alterations when compared with the other patients.

Total recording time with an SaO_2 of $\geq 95\%$ was slightly longer during N2 ($69.4 \pm 32.4\%$) and N4 ($73.0 \pm 26.3\%$) than during N3 ($61.5 \pm 31.0\%$), but this difference was not considered statistically significant. The recording time with an SaO_2 between 90% and 95% was $29.8 \pm 31.5\%$ for N2, $26.4 \pm 26.2\%$ for N4, and $35.9 \pm 27.3\%$ for N3 ($p = 0.093$; not significant [NS]).

Sleep Characteristics When Using Oxygen With or Without the DODS

An EEG recording for N3 was not available for one patient because of an EEG clock failure. Therefore, final polysomnographic results are presented for only 19 patients (Table 2). Regardless of the recording conditions, REM sleep time was below the 20% level usually found in non-COPD patients⁸: oxygen alone (N2), $12.3 \pm 8.7\%$; DODS + oxygen (N3), $16.4 \pm 7.8\%$; and oxygen alone (N4),

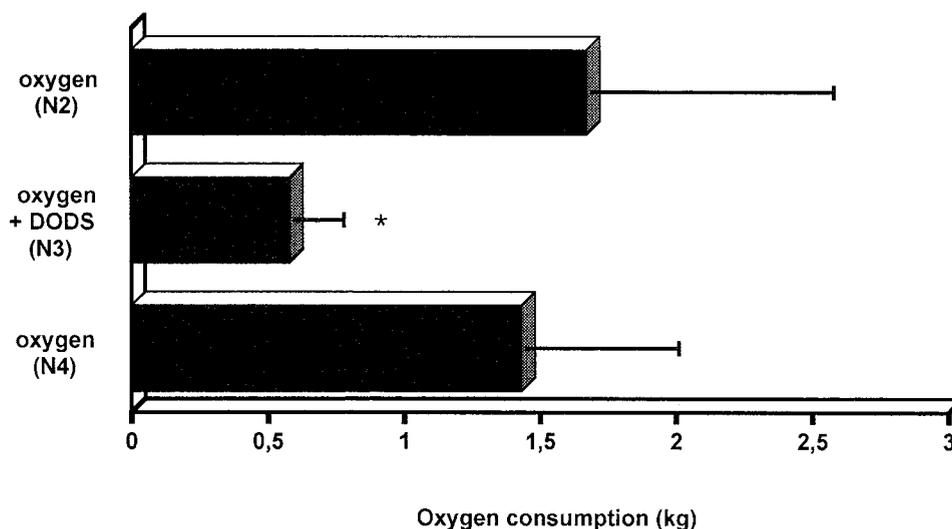


FIGURE 2. The mean amounts of oxygen consumed were identical for N2 and N4. Using a DODS device (N3) allowed highly significant savings (*, $p = 0.003$).

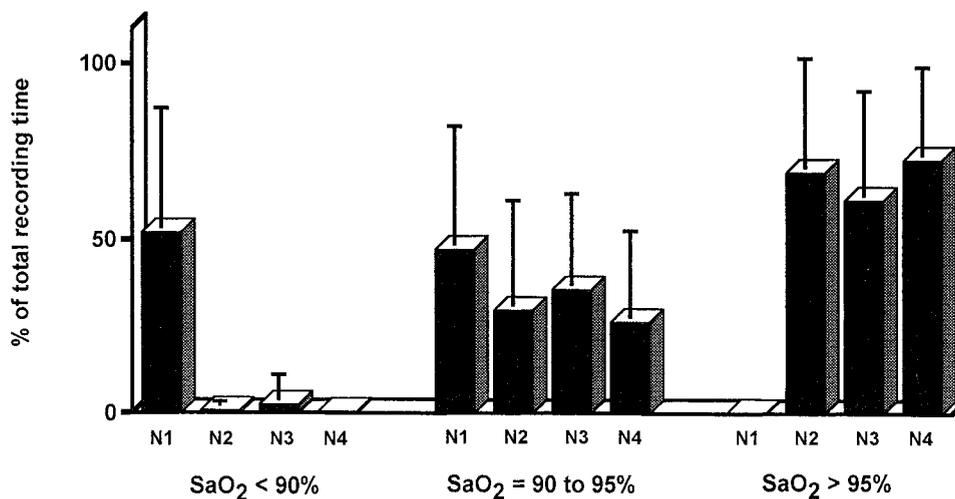


FIGURE 3. Nocturnal oxygen saturation values are expressed in percentage of total recording time. There were no statistical differences for any parameters between the two oxygen nights N2 and N4. Times spent at different SaO₂ levels were statistically similar between N2 and N3, and between N4 and N3 ($p = 0.093$).

15.3 ± 7.0%. The percentage of the night spent in NREM sleep was 83.7 ± 7.9% for N3 compared with 87.7 ± 8.7% for N2 and 84.7 ± 7.0% for N4 ($p > 0.05$). The percentage of stage 1/2 NREM sleep was 45.7 ± 14.2% during N3, 51.2 ± 16.4% during N2, and 49.6 ± 12.5% during N4 (NS). The percentage of stage 3/4 NREM sleep was 37.8 ± 14% for N3 vs 36.5 ± 13.1% for N2 and 35.1 ± 14.1% for N4 (NS) (Table 2). The patients spent slightly more time in REM and stage 3–4 NREM sleep during N3, and they woke up slightly less often, but the difference compared with N2 and N4 was NS.

In normal individuals, SOL is normally observed between 15 and 20 min. Mean values obtained from our COPD patients were always > 30 min but were not statistically different between the nights: 38.4 ± 33.5 min for N2, 52.8 ± 50.7 min for N3, and 38.2 ± 33.0 min for N4. REM SOL, normally found between 60 and 90 min, was 151.4 ± 101.6 min for N2, 109.0 ± 68.8 min for N3, and 110.2 ± 91.0 min for N4. The quantity of sleep assessed by the sleep efficiency index, which is normally ≥ 90%, was less than normal in this study, but no significant difference was observed between N3 and N2 or N4 (Table 2). The efficiency index was 69.6 ± 14.0%, with a mean number of arousals per night of 14.8 ± 6.9 for N3; 71.0 ± 15.3%, with a mean number of arousals of 16.4 ± 8.3 for N2; and 68.5 ± 15.0%, with a mean number of arousals of 16.1 ± 4.9 for N4. Patients 1 and 18, who had less satisfactory SaO₂ corrections while receiving oxygen + DODS when sleeping, did not have significantly different sleep parameters.

DISCUSSION

Respiratory Pattern During Sleep in COPD Patients With and Without the DODS

Sleep-related hypoxemia is a primary issue in managing severe COPD patients. Nocturnal desaturations are well related to the severity of diurnal SaO₂ at rest,^{8,9} but they cannot be reliably predicted from arterial oxygen desaturation during maximal exercise.¹⁰ In a vast majority of COPD patients, nocturnal hypoxemia is mainly related to hypoventilation during REM sleep because of a marked decrease of central respiratory drive and hypotonia of accessory respiratory muscles.⁸ Hypoventilation also occurs during NREM sleep, secondary to a decreased basal metabolic rate and an increased upper airways resistance.⁸ Additional ventilation/perfusion mismatch does occur during REM sleep,¹¹ but the extent of this phenomenon is not well known. Sleep apneas or hypopneas are triggering factors of nocturnal hypoxemia in only a minority of COPD patients. Sleep parameters are profoundly altered in COPD patients^{8,12–14} with a reduction of TST, REM sleep, and stage 3–4 NREM sleep. In contrast to reported studies,^{13,15,16} current research has demonstrated that sleep pattern and quality are improved by nocturnal oxygen therapy.^{8,17–19}

As previously mentioned, we observed a highly significant oxygen saving when using the DODS device (approximately 60%). Despite this oxygen saving, correction of nocturnal oxygenation (defined by the conventional SaO₂ threshold of ≥ 90%) was as effective as with oxygen alone, except for two patients. EEG records showed a marked reduction of

Table 2—Sleep Architecture*

	N3			p Value
	N2 (O ₂)	(O ₂ + DODS)	N4 (O ₂)	
REM sleep, %	12.3 ± 8.7	16.4 ± 7.8	15.3 ± 7	0.331
NREM sleep, %	87.7 ± 8.7	83.7 ± 7.9	84.7 ± 7	0.331
Stage 1-2, %	51.2 ± 16.4	45.7 ± 14.2	49.6 ± 12.5	0.15
Stage 3-4, %	36.5 ± 13.1	37.8 ± 14	35.1 ± 14.1	0.948
SOL, min	38.4 ± 33.5	52.8 ± 50.7	38.2 ± 33	0.563
REM latency, min	151.4 ± 101.6	109 ± 68.8	110.2 ± 91	0.144
Sleep efficiency, %	71 ± 15.3	69.6 ± 14	68.5 ± 15.	0.691
Arousals, No.	16.4 ± 8.3	14.8 ± 6.9	16.1 ± 4.9	0.654

*Values given as mean ± SD.

TST and sleep efficiency ($\leq 70\%$) with or without the DODS. The duration of wakefulness during sleep was higher than expected, with an average of 100 min, but not statistically different when using oxygen with or without the DODS. In addition to the underlying respiratory disease, the higher number of arousals and the increased REM and NREM sleep latencies were also responsible for the poor quality of sleep. Although none of these patients had obstructive apnea syndrome, the number of arousals was high (at least 14 per night), with no significant difference between nights. A higher mean value of SOL was observed when patients used oxygen + DODS (N3) than when they used oxygen alone (N2 or N4), and this was the only EEG parameter suggesting an alteration of sleep associated with DODS use; however, this difference was not statistically significant ($p = 0.563$). The constraints related to the protocol (early bedtimes and waking times) may have been responsible for lengthening of the sleep latencies and an artificial interruption of the morning sleep. Sleep staging revealed a higher percentage of stage 3–4 NREM sleep in our patients (37.8% with the DODS and 35.8% without the DODS) as compared with healthy adults (normally 25%). This observation is unusual in COPD patients in whom stage 3–4 NREM and REM sleep are commonly decreased and contribute to daytime sleepiness. In healthy adults, stage 3–4 NREM sleep is concentrated during the first half of the sleep cycle, and REM and stage 1–2 NREM sleep are observed during the second half. Stage 3–4 NREM sleep sometimes disappears from the last two cycles of sleep, which generally comprises four to six cycles. In our patients, a long SOL and an early awakening could possibly account for this abnormal sleep distribution, with a relatively large proportion of stage 3–4 NREM sleep. Moreover, age-related modifications of the various sleep stages should be taken into account²⁰; a reduction of stage 3–4 NREM sleep and a more homogeneous distribution of the various sleep stages during the night are often observed with

increasing age. Compared with healthy adults, our COPD patients had a decreased percentage of REM sleep but without any significant difference when either oxygen alone or oxygen + DODS was used.

Interest in DODS Use Among COPD Patients

Oxygen-conserving devices, such as reservoir nasal cannulas, transtracheal oxygen catheters, or the demand oxygen delivery valves, are effective ways to reduce oxygen costs and increase patient autonomy.^{4,6,21–25} The characteristic of a demand oxygen delivery valve is to deliver the gas at the beginning of each inspiration. In COPD patients breathing at a respiratory rate of 20/min and an inspiratory to expiratory ratio of 1 : 2, each respiratory cycle lasts 3 s with 1 s devoted to inspiration and 2 s devoted to expiration.²⁶ Only the gas inhaled during the first half of inspiration (representing two thirds of the tidal volume) participates in alveolar ventilation, the last third ventilating the dead space. Because only oxygen inhaled during the first 0.5 s of the respiratory cycle is used for oxygen exchange, oxygen therapy might be maximized by delivering the gas only during the first 0.5 s of inspiration.

A number of studies have shown that DODS devices ensure good quality oxygenation at rest^{4,6,26–29} or during exercise,^{28–32} although a recent study has cast some doubt on their ability to correct the most profound desaturations during exercise.³³ Indeed, very few studies have confirmed that such DODS devices do not influence quality of sleep and can be safely prescribed in COPD patients. **To our knowledge, only the study reported by Bower et al²⁸ has addressed this issue. These authors demonstrated the efficacy of DODS devices in maintaining SaO₂ during sleep in six hypoxemic COPD or restrictive patients.²⁸** However, a French study concerning two DODS devices (Optimox and COS5 [Nellcor Puritan Bennett]) reported unsatisfactory results for quality of nocturnal oxygen therapy among patients with chronic respiratory insufficiency.³⁴ These results were partly attributed by the authors to the absence of the apnea card with the Optimox valve. Furthermore, the COS5 valve equipped with the safety system triggered by apnea did not ensure more satisfactory SaO₂. These authors reported that the oxygen flow rate when using the DODS was equivalent to the flow rate delivered by continuous nasal oxygen therapy and was not previously adjusted individually according to the transcutaneous oxygen saturation as performed in our study. Indeed, the oxygen flow rate we used with DODS was adjusted to the patient requirements and was finally higher than the continuous oxygen flow rate (respectively, 3.0 ± 1.4 L/min and 2.4 ± 0.9 L/min). Similar con-

ditions were applied in the study conducted by Kerby et al⁶ among hospitalized patients.

Limits and Tolerance of the DODS Device

Challenging issues still persist concerning the tolerance of DODS devices. Patients may hear a slight clicking sound and may feel a small burst of oxygen associated with activation of the valve during inspiration. Some studies have adopted a subjective approach to this problem, and questionnaires revealed that patients often complain of the auditory discomfort at night.³⁴ Because of the Medilog system technology, our study may have overlooked the presence of microarousals from a clicking sound or a sudden burst of oxygen. However, such discomfort did not influence significantly the sleep parameters of our patients. A more serious difficulty may be linked to the changing respiratory pattern associated with nocturnal apneas or mouth breathing during sleep. This issue could not be addressed in our study because patients did not have any nocturnal apnea when breathing either room air or oxygen. As a safety factor, the apnea card appears essential to avoid interruptions of oxygen flow rate, and it may contribute to good oxygen saturation in patients with sleep apnea syndrome and COPD (overlap syndrome). In the study by Bower et al,²⁸ the oxygen-saving device sensed the negative inspiratory pressure whenever the patient was sleeping with open or closed mouth. Finally, use of the DODS device appears to be safe; unlike continuous oxygen therapy, pulsed oxygen therapy does not induce nasal dryness.^{4,35} Kerby et al⁶ estimated that the oxygen savings and humidifier costs would cover the expense of a DODS device within a 2-year period.

CONCLUSION

In summary, the DODS device ensures good quality oxygenation among a great majority of patients with moderate to severe COPD without sleep apnea syndrome and no significant alteration of nocturnal neurophysiologic profiles, provided that the oxygen flow rate is adjusted individually to a satisfactory transcutaneous SaO_2 . However, persistence of nocturnal desaturation in very few patients in our study still justifies providing a night of systematic ventilation polygraphy recording when prescribing a DODS device. Moreover, further studies are required to confirm the efficacy of this device in patients with an abnormal AHI.

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